CLINICAL AND EPIDEMIOLOGICAL STUDY OF CORROSIVE INGESTION

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CERTIFICATE

This to certify that the dissertation entitled "**CLINICAL AND EPIDEMIOLOGICAL STUDY OF CORROSIVE INGESTION** " is a bonafide original work of Dr. **K. SENTHIL MUTHU,** in partial fulfillment of the requirements for M.D. Branch – I (Internal Medicine) Examination of the Tamilnadu Dr.M.G.R Medical University to be held in March 2009.

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DECLARATION

I, **Dr. K. SENTHIL MUTHU,** solemnly declare that the dissertation titled, **"CLINICAL AND EPIDEMIOLOGICAL STUDY OF CORROSIVE INGESTION"** is a bonafide work done by me at Institute of Internal Medicine, Madras Medical College, during Jan 2008 to June 2008 under the guidance and supervision of Prof**. Dr. C. RAJENDIRAN, M.D.,** Institute of Internal Medicine. The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in Internal Medicine.

Place: Chennai.

Date: **(Dr. K. SENTHIL MUTHU)**

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CONTENTS

INTRODUCTION

Poisoning is one of the most commonly used suicidal methods. Caustic ingestion is one among them. In most of the poisoning mortality is observed in most instances. But once the life is saved immediately, the subsequent morbidity rarely witnessed. This is not so in caustic ingestion. Even though earlier mortality is very less, the morbidity in future is significantly high. Various studies conducted in the northern and southern parts of India documented significant exposure to caustic substances in both household and industrial products [51].

Many of the corrosive ingestions encountered in toxicological practice are used commonly in many households as well as occupational. Although most of the poisonings are intentional there are accidental poisonings as well because of its clear appearance and easy availability. Accidental ingestions are commonly encountered among children and psychiatric patients [3]. Caustic injury can produce devastating and progressive injury to esophagus and stomach [1,2].

Although most commonly affected body areas are the face, eyes, and extremities, all reported fatalities were as a result of ingestion. Little controversy exists regarding patient's management following dermal or

ocular caustic exposure. On the contrary, little has been proven in the way of medical treatment of caustic ingestion.

Ten percent of admissions in our Poison Control, Training and Research Centre, Government General Hospital, Chennai are due to caustic ingestion. There are very few studies regarding the epidemiological pattern of corrosive ingestion and on their follow up. Hence the present study.

AIMS & OBJECTIVES

- To find out the prevalence & pattern of Corrosive injury.
- To analyze the Age – Sex distribution among patients admitted with corrosive ingestion
- To elicit the circumstances for poisoning.
- To identify the common corrosives ingested
- To elicit the clinical features / clinical course and outcome
- To correlate the quantity ingested with the outcome
- To assess the grade of esophageal injury by means of upper GI scopy and correlate it with the outcome

REVIEW OF LITERATURE

INTRODUCTION

Caustic ingestion can produce a progressive and devastating injury to the esophagus and stomach [1,2]. Caustic injury remains a subject that is not as well studied as other esophageal and gastric diseases, even though knowledge of the proper approach to a patient with caustic injury can clearly make the difference between life and death.

In 2004, the "American Association of Poison Control Centre" toxic exposure surveillance system documented over 2 Lakhs exposures to caustic substances in both household and industrial products. Many of the corrosives encountered in toxicological practice are used commonly in many households as well as occupational. Although most of the poisonings are intentional there are accidental poisonings as well because of its appearance and easy availability. Accidental ingestions are commonly encountered among children and psychiatric patients [3].

Although most commonly affected body areas are the face, eyes, and extremities, all reported fatalities were as a result of ingestion. Little controversy exists regarding patients management following dermal or ocular caustic exposure. Immediate water irrigation of the site of

exposures, followed by routine burn care with analgesia, fluid and electrolyte replacement is the standard of care.

CAUSTIC AGENTS

There are two major types of caustic agents, namely

1 ACID

2 ALKALI

ACIDS

A Organic	-	Carbolic & Oxalic acids
B Inorganic	-	Hydrochloric acid, sulphuric acid & Nitric acid

PRODUCT TYPE	ACID INGREDIENT
Toilet bowl cleaners	Hydrochloric, sulphuric, Phosphoric acids & sodium Bisulfite
Metal cleaners	Hydrochloric acid

Pool cleaners	Hydrochloric acid
Antirust products	Hydrochloric, sulphuric & Hydrofluoric acids
Battery fluids	Sulphuric acids

ALKALIS

A. Sodium hydroxide

B. Potassium hydroxide

C. Lime

PRODUCT TYPE	CAUSTIC INGREDIENT
Drain cleaners	Sodium hydroxide, sodium Hypochlorite
Oven cleaners	Sodium hydroxide
Toilet bowl cleaners	Ammonium chloride
Household cleaners	Ammonium hydroxide, ammonium chloride

Bleach products	Sodium hypochlorite, Hydrogen peroxide
Dish washing detergents	Sodium carbonate, sodium Silicate
Button batteries	Sodium & potassium hydroxide

PATHOGENESIS

Even though acid and alkali have different pathogenesis for causing injury, the degree of injury in both depend [7,8] on

1. Agent
2. Concentration
3. Quantity
4. Physical state
5. Duration of exposure

Alkali causes more injury than acid, owing to its physical properties of being odourless and tasteless, it is ingested in larger quantity before the protective reflex is invoked. Also alkali has rapid tissue penetration.[1,9]

ALKALI

Alkali has a potent solvent action on the lipoprotein covering producing liquefactive necrosis with saponification of the mucosa, submucosa, muscularis of the esophagus and stomach [(1)]. It also causes thrombosis of adjacent vessels with further increase in necrosis. Exposure of 1ml of 3% NAOH for 1 second can penetrate the full thickness of esophagus as shown in experimental studies on rat.[(1)]

Acidity of the stomach does not protect against the alkali induced injury. If lye granules are taken orally, they will adhere to the oropharynx and cause more injury locally [(10)]. Button batteries cause injury by three mechanisms.[(11,12)]

1. Release of corrosive

2. Electrical injury

3. Pressure necrosis

ACID

Acid causes coagulative necrosis of the mucosa [5,6,13]. This causes less injury than the liquefactive necrosis caused by alkali because the coagulation formed will restrict the tissue penetration of the acid and hence further injury. Contrary to the earlier beliefs that stomach is injured more severely than esophagus due to the acidity and pyloric spasm [14,15], but animal experiments have shown that it is not so.

In both acid as well as alkali the initial life threatening injury may be perforation. If there is injury only in the form of ulceration, there will be sequelae in the form of stricture on healing.

Caustic exposure

⇩ seconds

Necrosis

⇩ 24 – 72 hrs

Ulceration

⇩ 14 – 21 days

Fibrosis

⇩ Weeks - years

Stricture

⇩ decades

carcinoma

Algorithm of the consequences of caustic injury as a function of time after ingestion

CLINICAL FEATURES

The initial presentation of the patient depends up on the time lag after ingestion, the amount and concentration of the corrosive. Most of the patients present with excessive salivation, chest pain, epigastric pain, drooling, edema of the oral mucosa, vomiting, hematemesis, dysphagia, odynophagia.[5,6,14,17]

If the injury is severe to the epiglottis and if associated with edema patient will present with stridor and hoarseness of voice. Some patients may be asymptomatic initially but may present with malena later. If the injury is very severe they may present with features of perforation and peritonitis. Tachypnoea, stridor and shock may be the initial presentation in a case of mediastinitis following severe injury to the esophagus.[18,19]

There are studies which have shown that there is no correlation between symptoms and degree of injury [20]. Hence, regardless of symptoms, especially with a history of ingestion of strong acid and alkali, it is mandatory to investigate thoroughly.[20]

Some patients may have progressive dysphagia followed by symptoms of gastric outlet obstruction like early satiety and emesis. In some patients there may be initial recovery and after 3 – 8 weeks they may present with symptoms of stricture. Some patients may progress to develop stenosis of the esophagus after 1 year.[21]

INVESTIGATIONS

Initial phase of investigations is aimed at history taking and symptom analysis. The ingestion may either be accidental or suicidal. If

the ingestion was accidental, the patient may attempt to spit out the corrosive as a immediate reflex, hence causing less injury.

On examination of the oral cavity, the injuries may vary from mild erythema to large necrotic ulcers. There will be mild to severe edema of the oral mucosa. A complete physical examination of the other systems has to be done to look for complications like aspiration.

Routine blood investigations have to be done which include a complete hemogram, renal parameters, serum electrolytes, arterial blood gas analysis to rule out metabolic acidosis. Chest X-ray should be taken to look for pneumo mediastinum and aspiration. If bowel perforation is suspected X-ray abdomen erect view may be taken. A CT abdomen with water soluble contrast may be required if X-ray inconclusive.[21,22,23]

Next important investigation is esophagogastroduodenoscopy (OGD) which is important in assessing the grade of injury.

Grade	Endoscopic Findings
I	Edema and erythema
IIA	Hemorrhage, erosions, blisters, ulcers with exudates
IIB	Circumferential ulceration
III	Multiple deep ulcers with brown, black, or gray discoloration*
IV	Perforation

Endoscopic Grade of Caustic injury

OGD should be performed in all stabilized patients without any features of perforation [6,7,24,25,26]. OGD should be performed within 48 – 72 hrs as the injuries are better defined (*kikendall et al)* [5]. At OGD scopy 40 – 80% of patients with history of caustic ingestion are found to have no injury. Injury with grade I, IIA rarely go for stricture but grades IIB, III 70 – 100% develop stricture.[10,16,28]

The role of barium contrast studies is only after 3 months to detect esophageal stricture or antral stenosis. Esophageal stricture can be of variable length with smooth or irregular margin. The thickness of the esophageal stricture can be measured with contrast CT.[29]

TREATMENT

For effective management of all acute poison victims (as common to all poisons) five complementary steps are required [51]. These are

1. Resuscitation and initial stabilization
2. Diagnosis of type of poison
3. Non specific therapy
4. Specific therapy
5. Supportive therapy

Resuscitation and initial stabilization

On arrival of a patient with corrosive poisoning, the initial steps are maintaining the airway, breathing and circulation. Since the corrosive poisoning patients are prone for laryngeal edema, the airway should be secured by endotracheal intubation if there is severe respiratory distress.

In cases with severe laryngeal edema, difficulties may be encountered during endotracheal intubation. Vitals should be stabilized.

NON SPECIFIC THERAPY

Unlike the management of other poisoning where gastric lavage forms the initial mainstay treatment, it is contra-indicated in corrosive poisoning due to increased risk of perforation.

Activated charcoal is not used as it does not adsorb the caustics and also interferes with OGD scopy findings. If perforation is suspected and confirmed by X ray/CT, surgical intervention should be done. Once the patient is stabilized, OGD should be performed at the earliest. However, a patient can be discharged without OGD if all the following criteria are fulfilled: (1) a detailed history can be obtained; (2) the ingestion was accidental, small in volume, and consisted of weak or low-concentration acidic or alkaline solutions; (3) the patient is asymptomatic and has no oropharyngeal injury; and (4) the patient can guarantee reliable follow-up if symptoms develop.

SPECIFIC THERAPY

The goal of therapy in caustic injury is

1 To prevent perforation

2 To avoid progressive fibrosis and stricture

UNPROVEN THERAPEUTIC MODALITIES

A. Neutralisation of caustics

If neutralization of caustic injury is tried there may be a rise in temperature in the gastric lumen due to exothermic reaction which will further increase the injury. But animal studies have shown no increase in temperature causing further injury. However, there is no human study to recommend this modality of treatment.[29,30,31,32]

B. Steroids

Usually after caustic injury, the natural course will be granulation tissue formation and fibro-elastic tissue reaction. Hence, if steroids are initiated within 24 hrs these inflammatory changes can be decreased, but there are no studies to favour this. Also, the steroids may mask the inflammatory features of mesdastinitis and peritonitis. So controlled studies are needed to determine the efficacy of steroid usage.[9,10,21,33,34]

C. Antibiotics

Local infection may increase the inflammatory reaction and granulation tissue. Hence, antibiotics may decrease this additional inflammation. However, no controlled studies are available to support its usage. [5,6,9,10]

D. H2 blockers and PPI

These two agents are given immediately to prevent the reflux, but there is increased risk of injury in case of alkali as the neutralizing acid may decrease. Hence they are recommended to be started after 24 hrs in case of alkali ingestion. No human studies are yet available.[39,40]

E. Nutrition

Parenteral nutrition has been recommended in patients with severe caustic ingestion to avoid damage to the esophagus or stomach that may occur if the patient is fed. There are no data to support this practice or to suggest that oral alimentation or tube feeding is injurious in acute caustic ingestion.

F. Early Esophageal Dilation.

Some authors have recommended the use of esophageal dilation immediately after injury. Dilation is performed at frequent intervals until healing occurs. There are no controlled data to support this approach, and others suggest that early dilation increases the risk of perforation and may accelerate fibrosis and stricture formation.[36]

G. Esophageal Stents

Endoscopically and surgically placed intraluminal stents have been used in patients with severe injuries to prevent and treat strictures. There are no long-term, controlled data to determine the efficacy of this approach [37,38]

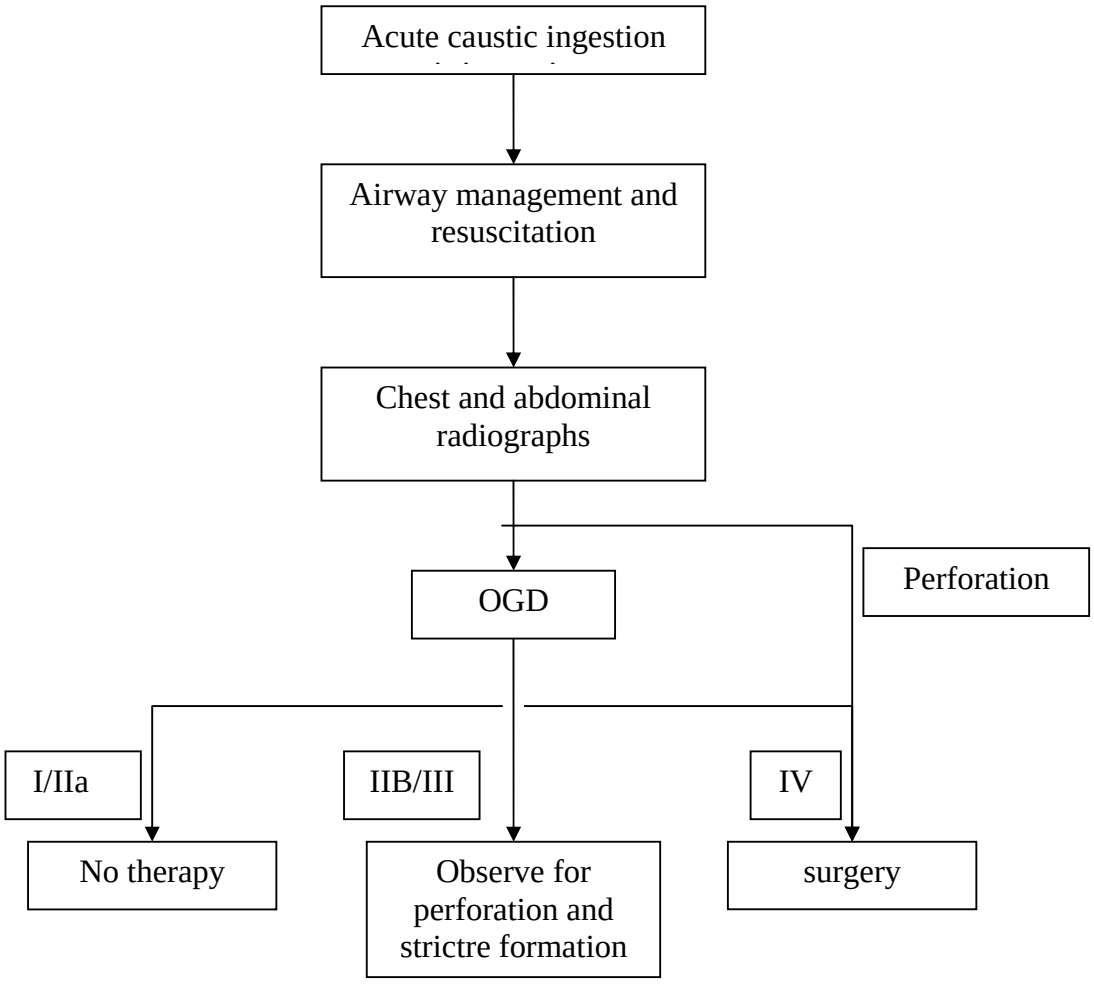
SUPPORTIVE THERAPY

In OGD scopy if the grade is I/IIA oral feeding with liquids can be initiated within 24 hrs and the chances of stricture formation in the future is low.

If the grade is IIB/III, the initiation of feeding depends on the patient condition. If there is no vomiting, no respiratory distress and if the patient is able to swallow saliva, oral feeding can be started after 48 hrs. if the patient is not able to swallow/tolerate oral feeding, feeding should be initiated through jejunostomy tube or naso-gastric tube.

During all these investigations and treatment, the vitals, acid-base and electrolyte monitoring should be done and any abnormality should be corrected. The patient can be discharged if they are able to maintain nutrition.

Barium swallow should be done routinely in grades IIB/III after 3 weeks to rule out any esophageal stricture or stenosis. Patients should be under follow up for 1 yr and if any symptom related to Upper GI tract like dysphagia should carefully investigated.[23]



Algorithm for the recommended approach to acute caustic injury.

LATE COMPLICATIONS

The late complications are:

1. Esophageal stricture
2. Antral stenosis
3. Carcinoma of esophagus

Esophageal Stricture:

Compared with other causes, caustic esophageal strictures require more numerous and more frequent dilations to achieve and maintain an adequate lumen. Care must be taken to dilate slowly and carefully. Perforation, bleeding, and sepsis may complicate dilation. Minor dilation-related perforations have been successfully treated without surgery using parenteral nutrition and antibiotics, with resumption of dilation in 4 to 6 weeks. Most per-forations occur in long, tight, eccentric strictures that have been dilated without fluoroscopic guidance. Proton pump inhibitors should be used as an adjunct to prevent acid reflux. Intralesional steroid injections at the time of endoscopic dilation have been reported to have a beneficial effect in an uncontrolled study. Rapidly developing, thick-walled strictures (as determined by CT scan) are more difficult to dilate and recur more rapidly. In 10% to 50% of patients with strictures, surgery

will be necessary because the patient cannot tolerate repeat dilation, nutrition cannot be maintained, or dilation is unsuccessful. When necessary, esophageal resection is performed with esophagogastric anastomosis or colonic or jejunal interposition.[41,42,43]

There are proposed methods for stricture prevention although they are not proven. They are

- Corticosteroids and antibiotics
- Antioxidants like vit. E (the O2 species formed after caustic injury are prevented and hence collagen synthesis)[44]
- Ketotifen, H1 blocker, mast cell stabilizers are proposed to decrease collagen synthesis [45]
- Phosphatidyl choline stimulates collagenases and prevents excess collagen accumulation [46]

ANTRAL STENOSIS

Antral stenosis after caustic injury usually develops in 1 to 6 weeks but may not appear for several years. It appears to be equally common in acid and alkali ingestions. Endoscopic dilation has been used successfully and should be considered as an initial maneuver in patients with antral

stenosis. Surgery may be needed and distal gastric resection is usually recommended. Although many patients are initially achlorhydric, vagotomy is usually performed along with antrectomy because acid production may return. Pyloroplasty and gastroenterostomy have been applied successfully in a few patients. With extensive injury, subtotal or total gastrectomy or partial esophagectomy may be necessary.[47]

CARCINOMA OF THE ESOPHAGUS

There is a strong association between caustic injury and squamous cell carcinoma of the esophagus. Between 1% and 7% of patients with carcinoma of the esophagus have a history of caustic ingestion. It has been estimated that there is a 1000-fold to 3000-fold increase in the expected incidence of esophageal carcinoma after caustic ingestion. Such a relationship is supported by the location of the cancer at the site of the stricture (scar carcinoma) and the younger ages of patients with caustic ingestion–related carcinomas. The average interval between injury and the development of squamous cell carcinoma is 40 years. Prognosis with combined surgical and radiation therapy appears to be somewhat better than that for other squamous cell carcinomas. This may be due to younger age patients who have developed earlier symptoms due to an already compromised lumen. Furthermore, the scar tissue may limit the spread of

the cancer. There are insufficient data to recommend surveillance EGD in asymptomatic patients with a history of remote caustic ingestion. Squamous metaplasia and carcinoma of the stomach have been reported after caustic injury. These reports are isolated, and there is no reported evidence of increased risk of development of gastric carcinoma in patients with previous caustic injury.[48,49,50]

MORTALITY

The mortality rate after caustic ingestion has decreased in the past 30 to 35 years from 20% to less than 1% as a result of lower concentrations of caustic solutions, improved surgical and anesthesiology techniques, and more effective antibiotics and nutritional support. However, even modern studies in some countries report mortality rates as high as 12% because of the high number of intentional ingestions. Most deaths result from mediastinitis, peritonitis, and subsequent multiorgan failure in individuals with third-degree injuries.[3,5,6,19]

MATERIALS & METHODS

The study population includes 60 consecutive cases of caustic ingestion, that were admitted in our Poison Control, Training and Research Centre, Government General Hospital, Chennai between January 2008 and June 2008. Patients were followed up prospectively at 3 months and 6 months after discharge.

Inclusion Criteria

The cases included were those with caustic ingestion alone

Exclusion Criteria

Mixed poisoning patients were excluded.

In our hospital all the patients with caustic ingestion were admitted in Poison Control, Training and Research Centre. As in all other poisoning, the initial step was to stabilize the vitals. A detailed history regarding the type and quantity of caustic ingested, the circumstances, presenting symptoms and details of first aid, if any were recorded. A thorough physical examination including the oral cavity was done. Then the patients were assessed for endoscopic procedures. If the patient was fit (Haemodynamic stable), the upper gastro intestinal endoscope was done within 48 hours. In the mean while routine blood investigation,

X-ray test were taken. Patients were kept nil per oral and supported with parenteral nutrition. In upper gastro intestinal endoscopy, there are 4 grades of injuries, from I – IV, in which Grade – II have a & b.

According to the grade of injury in upper gastro intestinal endoscope further planning was made. If the patients had less than Grade – II and were able to swallow the saliva, they were started on liquids with caution to avoid aspiration. If the injury was more than grade II, nasogastric tube was placed under endoscopic guidance and feeding was started through the NG tube. The patients were then transferred to the General Medical Ward where further stabilization of the patients both physically and mentally would be done. Every patient would be counseled separately and also with their family in medical ward as well as in Psychiatric OP Department. The patient would then be discharged after fixing date for barium swallow, usually after 12 weeks. Patients were discharged with nasogastric tube insitu for feeding. Patients were advised to follow up in Medical Gastro Enterology OP / concerned Medical OP / Review at Toxicological Department 211 on Thursday. In our study the patients were followed up at 3rd month and 6th month to assess the Grade of Dysphagia clinically and then subjected to barium swallow study. If the patients had any complications like stricture / gastric outlet obstruction further plan of treatment was discussed with the patient; usually endoscopic dilatation or surgery would be the modality of treatment.

OBSERVATION & RESULTS

In our study totally 60 cases of Corrosive Injury were admitted in the period from January 2008 to June 2008. This constituted 10% of total poison cases admitted in our Poison Control Centre and Research Centre, Government General Hospital, Chennai – 600 003.

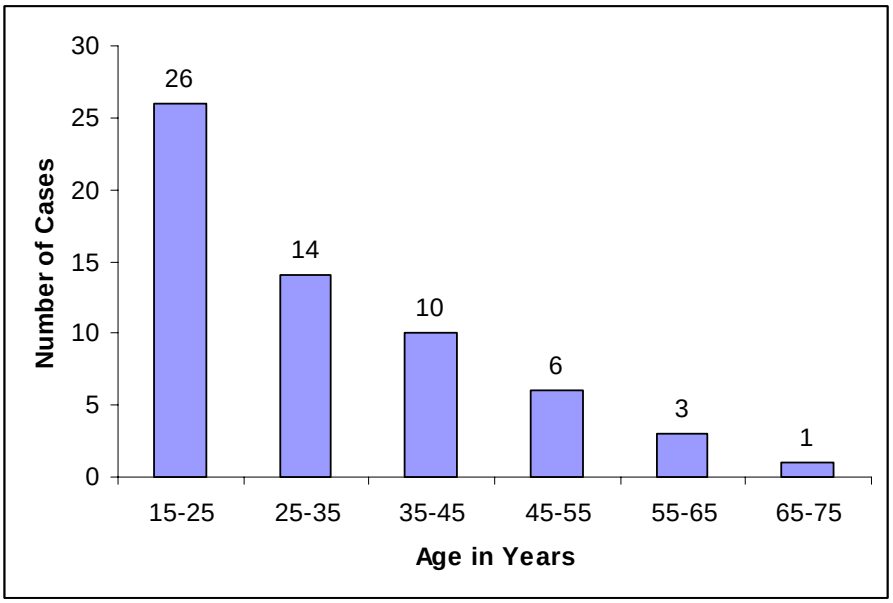
AGE DISTRIBUTION OF PATIENTS

The mean age was 29.5 years with range from 15 to 75 years in our study more number of cases were clustered between 15-25 years.

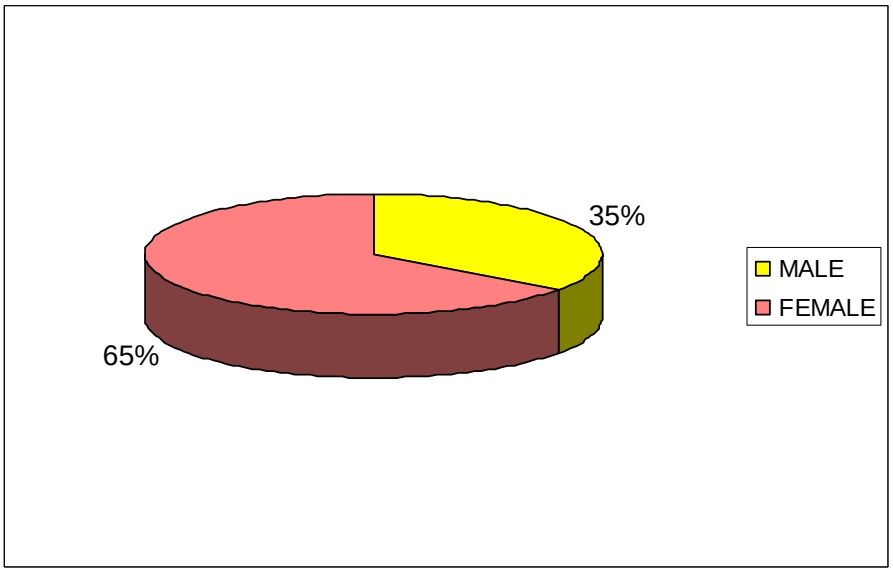
AGE DISTRIBUTION

Age in years	No. Of cases (n=60)
15-25	26
25-35	14
35-45	10
45-55	6
55-65	3
65-75	1

AGE DISTRIBUTION



SEX DISTRIBUTION



SEX DISTRIBUTION

In our study 65% of patients were women and 35% were men. The female patients had out numbered the male patients in our study.

SEX DISTRIBUTION

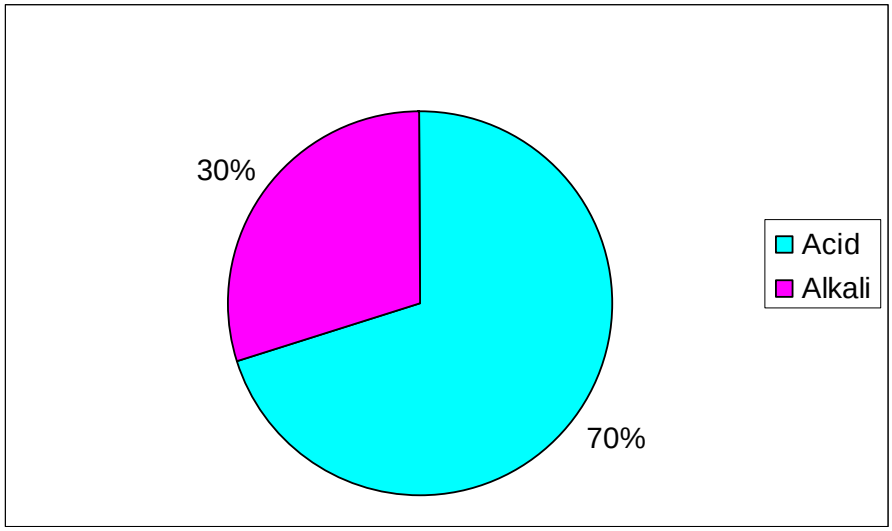
MALE	35%
FEMALE	65%

TYPE OF CORROSIVE

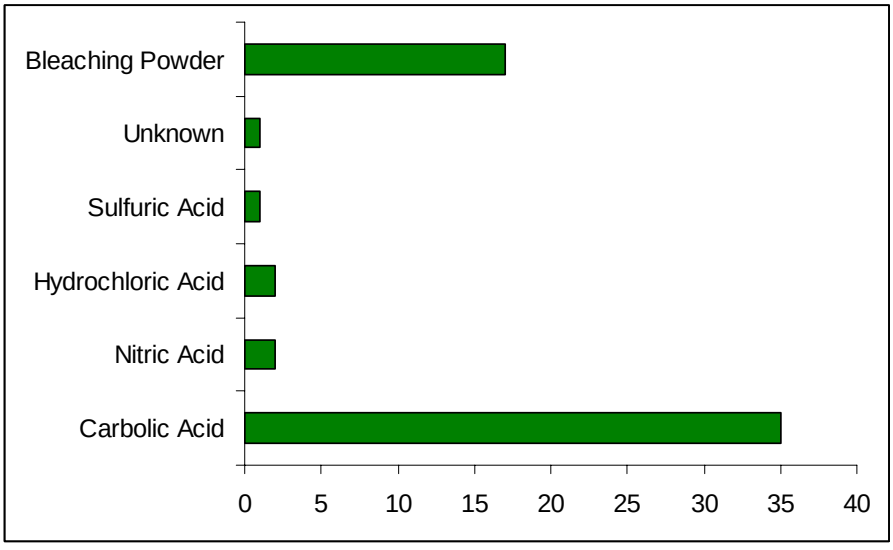
In our study the most commonly used corrosive was acid (70%), Alkali was only 30%. Among the acid, Carbolic Acid was the most commonly used acid. The most commonly used alkali was bleaching powder. The other acids used are sulphuric acid, nitric acid and hydrochloric acid.

TYPE OF CORROSIVE	NO. OF CASES
Acid	70%
Alkali	30%

TYPE OF CORROSIVE



SUB TYPE CORROSIVE



SUB TYPES OF CORROSIVE

CORROSIVE	NO. OF CASES
Carbolic Acid	35
Nitric Acid	2
Hydrochloric Acid	2
Sulfuric Acid	1
Unknown	1
Bleaching Powder	17

QUANTITY CONSUMED

The average quantity of corrosive consumed by the patients in our study was 50ml. The quantity ranged from 15ml – 100ml. The patients with more quantity had severe grade of injury in the UGI endoscopy.

In our study, suicidal intention was the most common circumstance under which the patient had consumed corrosive. This was observed in 93% of cases. Accidental consumption was noted only in 5% of our cases. Of which, three consumed at occupational site and two at home.

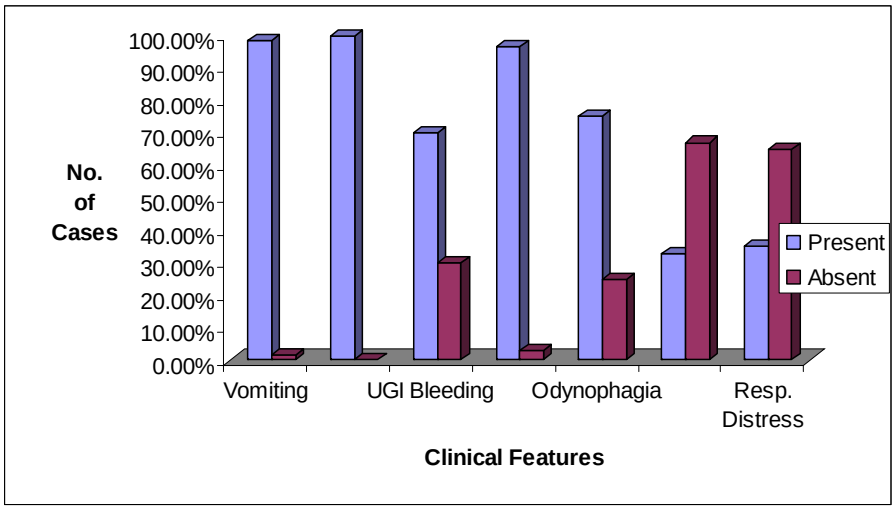
CIRCUMSTANCE	NO. OF CASES
Suicidal	92.80%
Accidental	7.20%

CLINICAL FEATURES

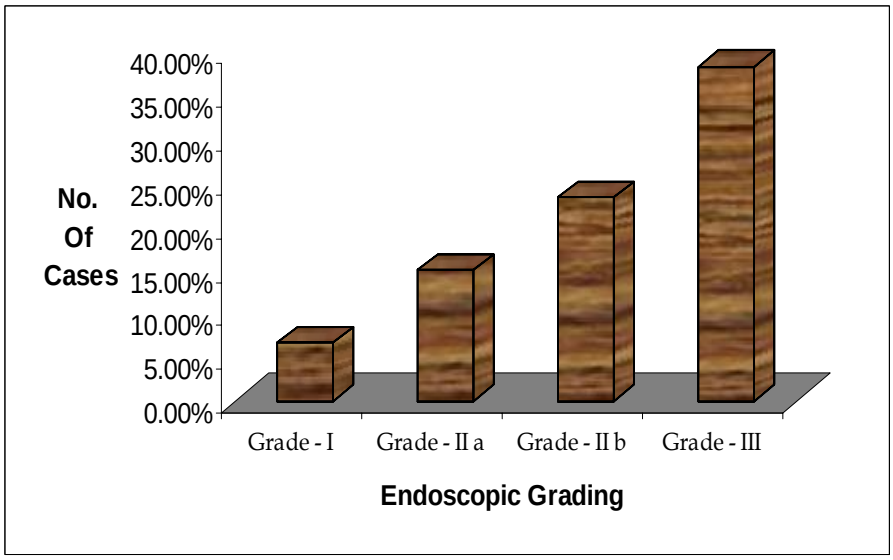
In our study out of 60 patients, 59 patients presented with vomiting out of which 42 patients had haemetemesis. Almost every patients presented with excessive persistent salivation. Out of 60 patients 22 patients had Respiratory Distress, among them 20 patients Stridor. Other common presentation was Dysphagia out of 60 patients 58 patients presented with Dysphagia among them 48 had Odynophagia. Abdominal pain was present in 54 patients.

Out of 60 patients, 9 patients presented with Glasgow Coma Scale of <7/15. All of these 9 patients had died.

CLINICAL FEATURES



UPPER GASTRO INTESTINAL ENDOSCOPY



Clinical features	N = 60 (%)
Vomiting	98.30%
Persistent Salivation	100%
UGI Bleeding	70%
Dysphagia	96.60%
Odynophagia	75%
Stridor	33%
Respiratory Distress	35%

INVESTIGATION

UPPER GASTRO INTESTINAL ENDOSCOPY

In our study, Upper GI endoscopy was done for 50 of our patients within 48 hours of hospitalization. The results were: Grade –III injury was present in 38.3%, Grade – IIa 15%, Grade – II b 23.3% and Grade - I 4%. For 10 patients, the UGI Scopy was not done due to poor haemodynamic state.

ENDOSCOPIC GRADING	NO. OF CASES
Grade - I	6.60%
Grade - II a	15%
Grade - II b	23.30%
Grade - III	38.30%

DURATION OF HOSPITAL STAY

The mean duration of hospital stay for Caustic Injury Patients was 6.5 days ranging from 3 -15 days. This stay included the initial toxicology IMCU admission and further stay in general ward.

MORTALITY RATE

Out of 60 patients, 48 patients were discharged. 9 patients died within 48 hours. Out of these 9 patients, 5 patients Forensic Report showed perforation of the stomach. All these patients were admitted with Glasgow Coma Scale < 7/15.

MORTALITY DATA	NO. OF CASES
Discharged	80%
Death	15%
AMA	5%

FOLLOW-UP

In our study we planned to have follow up of the patients every 15 days and then to assess the grade of dysphagia at 3rd and 6th month. Dysphagia was graded using Ankinson Scale.

At 3rd month we had follow up of 37 patients (77%), out of 48 patients, 11 patients were lost to follow up. Out of these 37 patients, 13 had dysphagia of Grade – III. i.e., Dysphagia for soft food. 11 had Dysphagia of Grade IV i.e. Dysphagia for liquids. 13 Patients had No Dysphagia.

GRADE OF DYSPHAGIA	**NO. OF CASES**
Grade - III	35%
Grade - IV	30%
Nil	35%

For all the above patients who had dysphagia, Barium swallow study was done, which showed following pattern of stricture pattern. 13 patients had Stricture of Oesophagus and 2 patients had Antral Stricture. Among the 13 patients who had Oesophagial Stricture, 2 patients had Antral Stricture also.

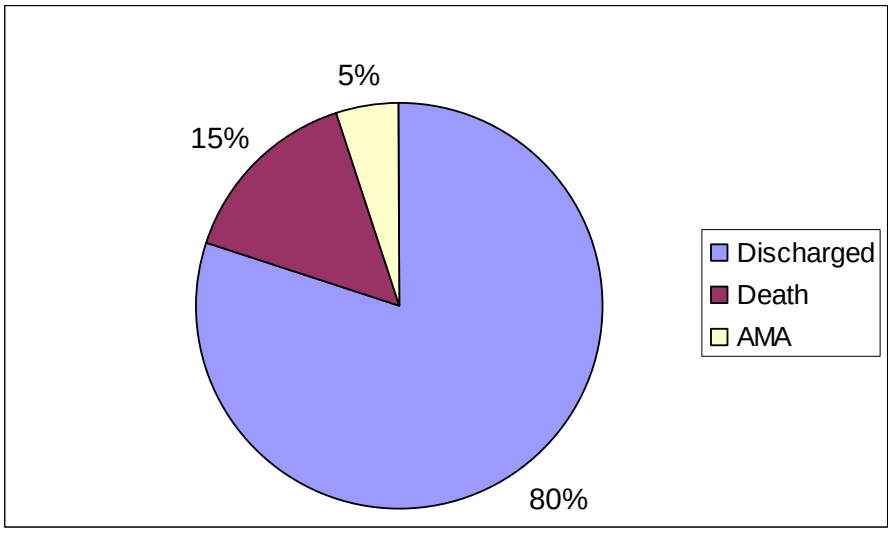
STRICTURE PATTERN	**NO. OF CASES**
Oesophagus	13
Antral Stricture	2
Both	2
Normal	23

TREATMENT OF COMPLICATION

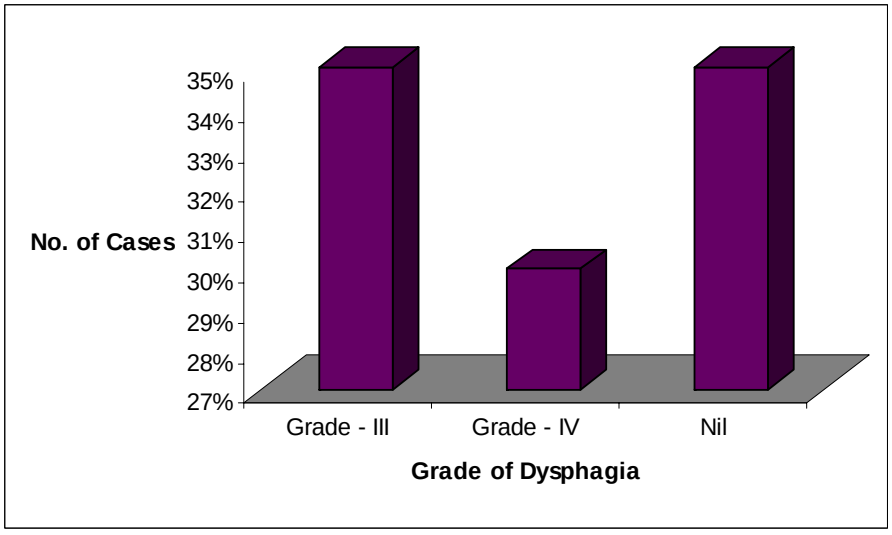
In our study out of 13 patients, 11 underwent endoscopic stricture dilatation and 4 had surgery. Antrectomy was done in 2 patients and pharyngocoloplasty was done in remaining two of our patients.

PROCEDURES	**NO. OF CASES**
Endoscopic Dilatation	11
Surgery	4

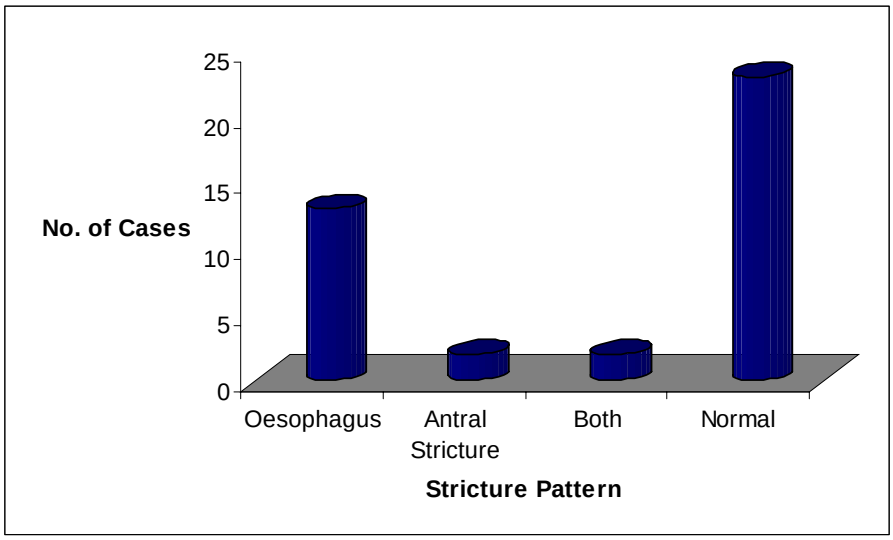
MORTALITY RATE



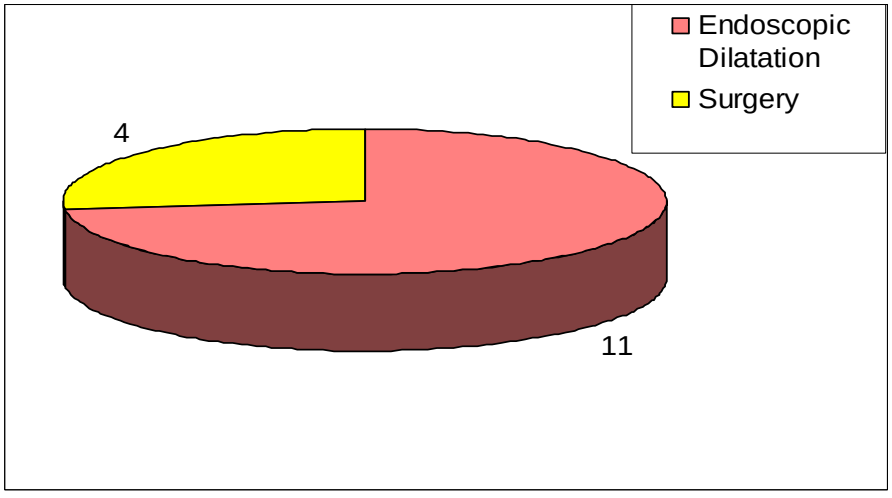
GRADE OF DYSPHAGIA



STRICTURE PATTERN



TREATMENT OF COMPLICATION



DISCUSSION

Caustic ingestion can produce devastating injury to the upper digestive tract, damage in rapid, its results potentially catastrophic and its complications if the patients surveys, may last a life time. Further more, little has been proven in the way of medical treatment, short of surgical section, to alter the course of injury. Nonetheless caustic injury remains a subject that is not as well studied as other oesophageal and gastric diseases, even though knowledge of the proper approach to a patient with caustic injury can clearly make the difference between life and death.

In our Poison Control, Training and Research Centre, Government General Hospital, Chennai, 10% of poison cases admitted were Caustic Injuries. We had taken a Socio Demographic Study of Caustic Study which include epidemiological, clinical, and with investigation like U.G.I. Scopy and further follow up of the patients.

The mean age group of the patients in our study were 29.5 years with range from 15 – 75. The study conducted by *broor et al* included totally 138 patients (over the period of 5 years). With mean age of 47 ranging from 14-97. Comparing the study the patients in our study group were younger and in the productive group.

Study published by *Eliasher R et al* consist of totally 50 cases. The age group range from 5 months to 71 years. But in our study paediatric age group was not included. In 2003, Study conducted by *Shivakumar et al* with totally 30 patients was published. The mean age group was 28.5 years, which was nearly equal to our study.

The common age group of Caustic Injury in our study group was between 15 – 25 years (26 patients).

In our study 65% of Female Patients and 35% of Male Patients had consumed Corrosives. Female patients had outnumbered the male patients. But in the study by *Broor et al* Male patients (84) had outnumbered the Female patients (74). Compared to our study the study conducted in Turkey (September 2004) also had more number of Female Patients.

Acid (70%) was the most common caustic consumed in our study. Only 30% had consumed Alkali. While in the most of the Western World Studies (MC. Guigan MA, et al., Jungler DJ, et al) Alkali was the most common type of corrosive consumed. The *Eliasher R et al* study also showed Alkali as the most common type consumed than acid. The same result was shown in Spanish Study.

But the studies conducted in Turkey 2004 had showed Acid as the most common type which was compared to our study.

This variation of Western World with more Alkali consumption was attributed to the wide range of products that contain Alkali while in the East Corrosive Acids are readily available in the form of Toilet Bowl Cleaners. Hence more Acid injuries are recorded here.

In our study Carbolic Acid was the most common Acid Consumed. This was due to easy availability of Toilet cleaner with carbolic acid. In Spanish Study Nitric Acid was the most common acid type consumed. Where as in the Netherland Study Glacial Acid was the most common type. The study conducted by *Gupta et al* showed Sulphuric Acid as the most common type. So the most common sub type of acid consumption depend upon the local availability of the particular subtype.

Bleaching Powder (Sodium Hypochloride) was the most common Alkali consumed in our study. All the patients with alkali injury in our study had consumed only sodium hypochloride. In *Jougland J et al* study, sodium hydroxide was the most common alkali consumed.

In our study Suicidal was more common than accidental ingestion. Most of the study from Western World likes the Spanish Study (Suicidal 34%, Accidental 51%), Laryngoscope 2006 August (Accidental 67%, Suicidal 33%), Turkey 2004 September (Accidental 54%, Suicidal 36%), and Quotes Accidental Consumption more than Suicidal. Compared to our study, the study conducted in Stanley Medical College 2002 also showed Suicidal was more than accidental.

About 98.3% of patients presented with vomiting, out of these 70% had haematemesis. In the study conducted by *Shivakumar et al*, 56% of patients presented with UGI Bleed, 96% presented with Dysphagia. Almost all patients presented with persistent salivation. 33% presented with Respiratory Distress and Stridor. Compared to our study the Stridor percentage (50%) was more in the study conducted by *Shivakumar et al*.

On Clinical Examination of the oral cavity almost all patients had inflammatory change which range from mild erythema to ulcer.

The mean amount of corrosive the patient had taken was around 50ml. The range varies from 15-100ml. The amount of corrosive consumed played significant role in the injury of the upper Gastro Intestinal Tract.

In upper gastro intestinal endoscopy above 40% had Grade – III Injury. 25% had Grade – II Injury. 15% had Grade – IIa and 7% had Grade - I. More or less same percentage of different grades of injury was noted in study conducted by *Shivakumar et al*.. Whereas study conducted by *Gupta et al*, out of 15 patients, above 10 patients had Grade–III Injury. In Spanish study Grade – I was more than Grade – II & Grade – III.

In our study Stomach was involved in 40% of the patients. All the Grade – III injured patients had stomach involvement. Duodenum involvement was noticed only in 20%. In spanish study also the same percentage of stomach and duodenum involvement was noticed. But, Grade – III injury in this study was only 13%.

The mean duration of the hospital stay of the caustic ingested patients were 6.5days. The duration of hospital stay varies from 3-15 days. This duration of stay includes the initial toxicological IMCU stay also. In study conducted in Netherland 2004, the mean duration of the hospital stay was 7.2 days. But in their study only 49% of patients were admitted in IMCU, the rest of the patients were managed in General Ward.

The mortality rate in our study was 15%. Out of 60 patients admitted, 9 patients were died. All these patients were admitted with Glasgow Coma Scale < 7/15. The Forensic Report of 6 patients had showed the perforation in the stomach. In 3 patients the stomach had torn in two pieces. The mortality rate of study done by *Shivakumar et al* was 25%. Whereas the mortality rate of Spanish Study was 5.7%.

Martal et al., showed the fall in mortality rate from 20% to 1%. They have attributed this fall in mortality rate as a result of lower concentration of caustic solution, improved surgical and anaesthetic technique and more effective antibiotic and nutritional support. But, this was noticed only in accidental ingestion group. *AZZOLINE et al*., showed the mortality rate was around 12% in the patient who had taken corrosive intentionally.

In our study we had follow up of 61% of patients. We had follow up at 3^{rd} and 6^{th} months. Out of 37 patients, 13 patients had Grade – III Dysphagia, 11 patients had Grade – IV Dysphagia, 13 patients had Grade – I Dysphagia. For all these patients barium swallow study was done. Out of 37 patients, 13 patients had Oesophageal Stricture, 2 patients had

Antral stricture, 2 patients had both. In Spanish study only 5% patients had Oesophageal Stricture. Those patients who had developed Oesophageal Stricture had Grade – III injury in the upper gastro intestinal endoscope. In *Broor SC et al*., the oesophageal stricture was seen in 13 out of 36 patients.

In our study the oesophageal stricture patients were treated with endoscopic dilatation. In 4 patients surgery was done, for 2 patients Pharyngocoloplastic was done and for others 2 antrectomy.

CONCLUSIONS & SUMMARY

CONCLUSIONS

- 10% of poison cases admitted in Poison Control Centre and Research Centre, Government General Hospital, Chennai, were Caustic Ingested patients.
- Females had outnumbered the males in our study. (65:35%)
- The common age group was between 15 - 25 years (26 out of 60 Cases).
- Acid was the most common corrosive consumed in our population.
- Suicidal ingestion was more common than accidental in our study group.
- The quantity of caustic consumed determined the outcome.
- Patient with Grade – III injury had more morbidity in the form of stricture involving upper gastro intestinal tract.
- 100% mortality observed in Grade – IV injury.

SUMMARY

In India corrosive poisoning is a common method to inflict self harm. The unfortunate aspect is that even if the patient tides over the acute crisis, he is left with a significant complication in the form of Oesophageal strictures. These strictures significantly affect the quality of life and severely handicap the patient. The patients are unable to enjoy even simple pleasures in life like Food and water.

The underlying reason for the patient to consume the corrosives are usually trivial and evanescent, however they are left with a handicap which slowly but definitely starves the patient to death.

According to my study, the grade of Oesophageal injury is directly proportional to the amount of corrosive ingestion and the concentration of the Corrosives. Hence it is imperative that, to prevent the occurrence of this crippling & painful disorder, drastic measures need to be adopted. This should include Public education, Legislative measures & health care measures.

Public education towards the hazards of corrosive injury should be undertaken; they should be educated towards safe and secure storage of the corrosives.

Legislative measures in the form of proper labeling and packing, Prominent Warning symbol on the Containers, etc. we can pass Laws which restrict the concentration of the Corrosives to less than 10% as done in Western countries. This may not prevent the poisonings but it will definitely go a long way in reducing the grade of Oesophageal injury and the subsequent stricture formation.

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ANNEXURES

Clinical study on Corrosive injury in adults

PROFORMA

Case No.

Name: Age/Sex: IP No:

Address: Ph. No.

Date time: where consumed where recognized

Prehospital treatment :where when what

Presenting complaints:

Time lapse since consumption:

Type of corrosive : Acid/ Alkali Compound:

Percent / Conc.: Quantity:

Empty stomach/ after food

Suicidal/ Accidental / Homicidal

Vomiting : Yes/ No Duration: days

Onset : Rapid/ Insidious

Induced vomiting: Yes/ No

Hematemesis : Yes/ No

Dysphagia : Yes/ No Solids/ semi solids/ liquids/ saliva

Odynophagia : Yes/ No

Drooling of saliva : Yes/ No

Stridor/ Hoarseness : Yes/ No

Chest pain : Yes/ No

Breathlessness : Yes/ No

Abdominal Pain : location: nature:

Aggravating/ relieving

Abdominal distension : Yes/ No

Malena : Yes/ No

<u>Past History</u> :

Peptic ulcer disease/ chronic NSAID use

Abdominal Surgeries : Yes/ No

Alcohol intake : Duration: Years Amount: ml/day

Smoking : Yes/ No Duration:

<u>Examination</u> :

Conscious : Others :

Oral cavity : Lips/ Tongue/ Pharynx: ulceration/ edema/ exudates:

PR: / RR: BP:

Abdomen:

Distension :

Tenderness : Present/ Absent Location: Severity: mild/ mod/ sev

Hepatomegaly: Yes/ No Size:

Splenomegaly: Yes/ No Size:

Free fluid : Yes/ No. If Yes: Minimal/ Moderate

Complication:dehydration sepsis shock

Investigations:

Hb: g% TC: DC: N/L/E:

Platelet count: ESR:

Blood urea mg% Blood Sugar: mg%

<u>LFT</u>

Bilirubin mg% ; AST IU/L; ALT IU/L

SAP U/L; Protein (T):

Chest X-ray:Mediastinitis/ Pulmonary Infiltrate / subcutaneous emphysema/ a

X-ray Abdomen: Air under the diaphragm/ dilated bowel loops

Others:

Endoscopy:

Esophagus:

Grade	Endoscopic Findings
I	Edema and erythema
IIA	Hemorrhage, erosions, blisters, ulcers with exudate
IIB	Circumferential ulceration
III	Multiple deep ulcers with brown, black, or gray discoloration*
IV	Perforation

Extend of involvement:

Stomach: edema/ erosion/ hemorrhage/ ulcerations/ exudates

Fundus:

Body:

Antrum:

Pylorus:

Duodenum:

Bulb :

D2 :

Treatment

IV fluids; NPO - days; PPI/ Ranitidine; Steroids (if any)

Antibiotics:

Initiation of nutrition: on Hrs/ Day

Any therapeutic intervention done: Yes/ No

Duration of hospitalization: ICU: Total:

Outcome : Discharge/ AMA/ Death

At discharge

Grade of dysphagia at discharge

Feeding Jejunostomy/ RT feeding

FOLLOW UP

Visits :

Duration :

Symptoms:

Dysphagia Grade :

Early satiety :

Weight loss :

Progressive emesis :

Signs:

Features of GOO :

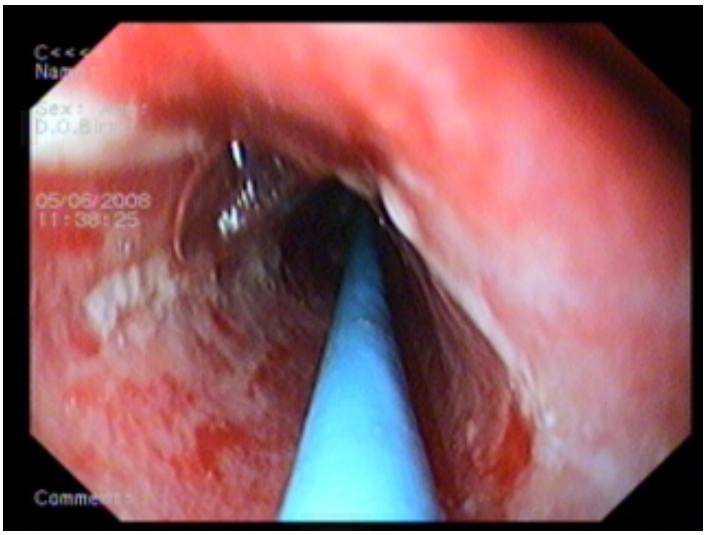
Barium Swallow (3 wks later) :

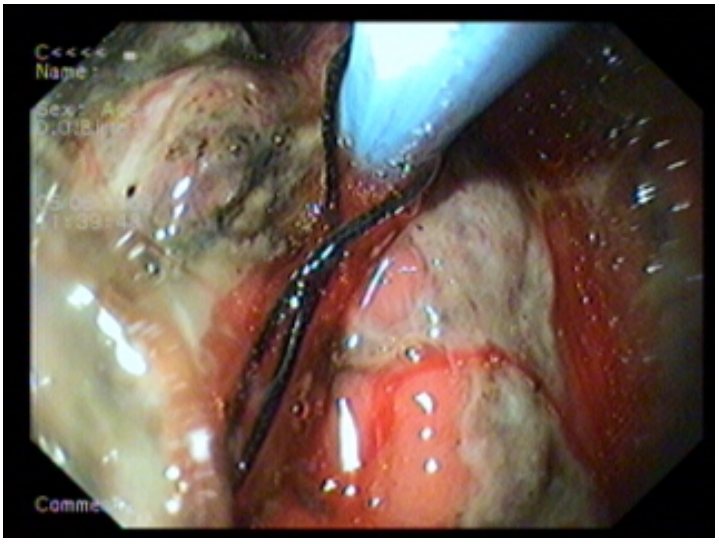
OGD (if any) :

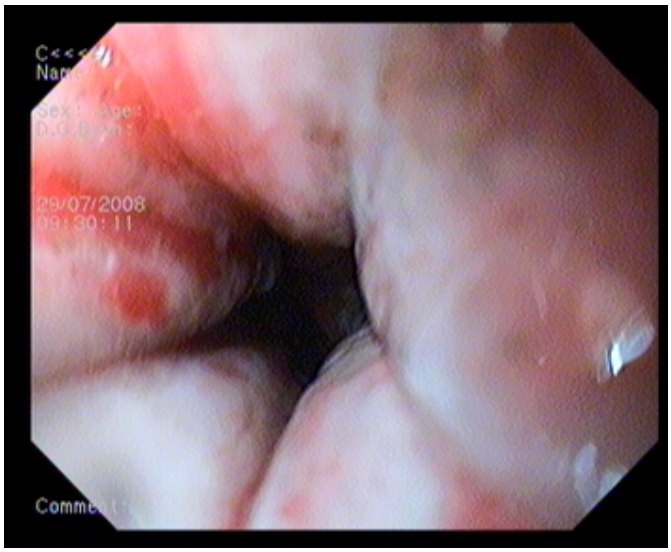
Dilatation (if any) :

Surgical procedure :

NASOGASTRIC TUBE INSERTION IN A PATIENT WITH CORROSIVE INJURY







GRADE II A INJURY